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# CLINICAL AND PATHOLOGICAL OBSERVATIONS ON CASES OF INJURY OF THE CERVICAL SPINAL CORD.[1]

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THIS paper is designed to call attention to some of the more obscure conditions that are observed in cases of injury to the cervical portion of the spinal cord. The cases on which the paper is based are five in number. Of these, four occurred in the Presbyterian Hospital in the service of Dr. Chas. K. Briddon, to whom I am much indebted for the opportunity to study them.

CASE I.—The patient, J. R., was a laborer, 30 years of age. There is no family history of interest, except that his father died from a broken neck. The patient himself is well built, and has no recognizable disease, but is a confirmed alcoholic.

On the evening of November 20th, about 9 o'clock, while drunk, he fell headlong into a cellar-way, a distance of ten feet, and soon after was taken to a police-station, where he spent the night. Next morning his intelligence was clear, but he was quite unable to move, and was soon taken to the hospital.

*Physical Examination, Nov. 21st*—Heart and lungs normal. Pulse 50. Respiration shallow and slow, and almost wholly diaphragmatic. No dyspnœa. Temp. 98°. There is great tenderness over the cervical spine. In the pharynx is an exceedingly prominent angle, probably the body of the third cervical vertebra. On cautiously moving the head laterally, and on rotation, there is distinct crepitus in the lower cervical region

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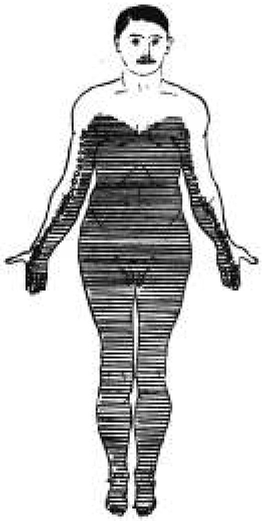
[1] Read before the New York Neurological Society, March 3d, 1891.

As the patient lies in bed, the forearms are flexed and the arms abducted. The fingers are somewhat flexed and the hand lies upon the chest, the fingers coming near the median line, and within three or four inches of the inner end of the clavicles. The upper extremities show a strong tendency to maintain this position; when the arms are extended by the side, they resume their original position. Below the arms the patient is completely paraplegic. In the arms the state of muscular power is as follows: Right arm, flexors and extensors of fingers and interossei completely paralyzed; flexors of wrist very paretic; left arm, flexors and extensors of fingers and interossei very weak; flexors of wrist slightly paretic. The extensors of the wrist, the biceps, triceps, and supinators and pronators and deltoids are normal in power on both sides. The biceps muscles are in a state of contracture, but the contracture can be temporarily overcome by moderate traction. When this contracture is overcome by extension, the biceps muscle becomes the seat of widespread fibrillary contractions, which continue many seconds after the extension is accomplished.

The condition of sensibility is indicated in the accompanying diagram, and requires no minute description. The loss of sensibility is to pain, touch, temperature and muscular sense, but the patient is conscious of a sensation of cold over the entire body. The pain in the outer aspect of the arm is considerable, and is greatest in the forearm and thumb. Sometimes the sensation is of an intense burning character. These irritative sensory phenomena are constant, but vary in degree as well as in character.

The knee-jerks were absent when the patient was admitted. The superficial reflexes were also abolished, cremasters, plantars, abdominals. The patient passes urine and fæces involuntarily. There is distinct priapism toward evening.

The patient's intelligence has been clear since admission. Temperature at 12 P.M. 105°. Pulse 60. On morning of Nov. 22d (day after admission), temperature 104°. Intelligence clear. Breathing diaphragmatic. The extraordinary muscles of respiration are acting more strongly than yesterday. Examination on evening of the 22d gives essentially the same results as that of Nov. 23d (10 A.M.), namely, the arms maintain a position similar to that already noted. The forearms are now supinated and elevated from the chest to the sides of the head; the arms are abducted. The extensors and flexors of the wrists are completely paralyzed; pronation is feeble, but supination is good; the triceps is weak on either side; biceps and deltoids normal. Rotation of arm perfect. No ascent in the area of anæsthesia. The upper limit of the anæsthesia and analgesia coincide closely. Irritative symptoms have ceased. There is no pain or hyperalgesia along outer border of arm and fingers. There is no muscular rigidity in biceps. No myoidema has been noted at any time.



Reflexes are absent. Priapism is marked. During the evening of the 23d the following observations were made: The only change in muscular power since morning is that both biceps are a little weak. Limits of anæsthesia same as before. Pupils equal, a little contracted, react to light and during accommodation. The left palpebral fissure seems a little smaller than the right, which is of normal size. Reflexes absent as before. Priapism has passed away. Slight irritation of the skin at any part, especially near the genitals, causes an immediate erection of the penis, which lasts for a few seconds only. Highest temperature for the day $103\frac{5}{10}$°.

*Nov. 24th.*—Patient's general condition fair during morning, and poor toward evening. Highest temperature during day is 106.5°, at 9 P.M., when the pulse was rapid and feeble. The loss of power in the biceps muscles is greater than on the day previous, and there is commencing weakness of the deltoids. Pronation and supination are possible, but both movements are feeble. The limit of anæsthesia on the chest is practically unchanged. On the arms and fingers the loss of sensation has extended outward (cephalad) slightly, perhaps one-third inch. Priapism has returned. The pupils are unchanged. The cremaster reflex has reappeared.

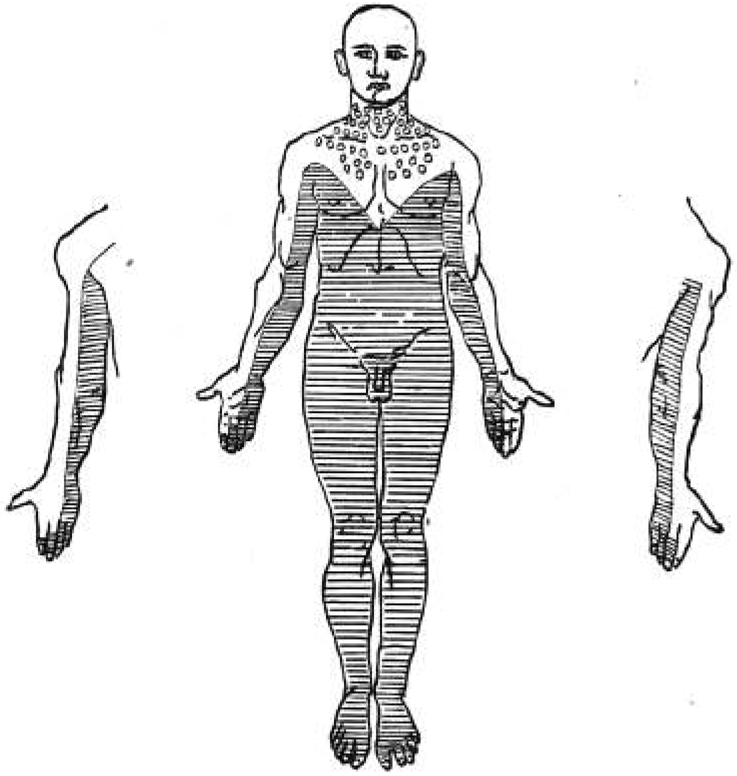
*Nov. 25th.*—Temperature remains high (highest 106°), and patient is beginning to fail. Respiration 20, irregular in rhythm and force. Pulse 75, fair strength, slightly irregular. Sensibility cannot be accurately tested, because patient is mentally clouded, but there is probably no extension upward. The biceps muscles are weaker than yesterday. Pronators and supinators not distinctly weaker than before. Cremasteric reflex is present. There is no priapism. The pupils are smaller than they have been, and react only slightly to light. There is no distinct evidence of atrophy in the muscles of hand and forearm. Electrical examination gives the following results: Faradic irritability is greatly diminished in all the intrinsic muscles of either hand. There is probably some diminution in faradic irritability in the flexors and extensors of the wrist, but this is uncertain. Galvanic irritability is lost in the intrinsic muscles of the hand, with the exception of the first dorsal interosseus of the right side, which reacts feebly, and shows KCC=ACC. The flexors of either side show slightly diminished galvanic irritability, without reversal formula.

*Nov. 26th.*—The patient was in stupor during the early hours. Temperature fell from 106° to normal. The features are pinched, pupils contracted and eyes sunken. Pulse is small and feeble. An examination made at 9 P.M. shows no change in the distribution of the motor and sensory paralysis from that of yesterday. The reflexes are absent as before. An electrical examination of the muscles of the left arm show entire loss of faradic irritability in the intrinsic muscles of the hand, and distinct diminution in that of the flexors and extensors of the wrist. The supinator longus and pronator radii teres show no changes in irritability. There is complete loss of galvanic irritability in the hand muscles, and distinct diminution of irritability, with sluggish contraction, but without reversal formula, in the flexors and extensors of the wrist.

*Nov. 27th.*—The patient sank rapidly, and died at 10.30 A.M. of respiratory failure.

The autopsy, made fourteen hours after death, showed a dislocation forward of the sixth cervical vertebra upon the seventh, and fracture of the lamina of the seventh on the right side. There was also found a fracture of the lamina of the second vertebra on the left side. As the dura lay exposed at the level of the dislocation it was normal in appearance, and its contents seemed of normal consistence. On opening the dura, however, the cord was found to be very much softened at the level of the eighth cervical segment, and somewhat reduced in consistence in the seventh segment. At the level of the seventh segment the central canal was slightly distended with blood. The microscopical examination gave the following results: In the eighth cervical segment the topography of the gray and white substance was scarcely recognizable, although the remains of the gray could, for the most part, be readily distinguished from that of the white. Anteriorly, on either side of the cord, was an irregular gray mass containing a few shrunken bodies that appeared to be the remains of ganglion cells. The shrunken and structureless remains of the posterior horns could readily be made out. The gray commissures were a narrow line of *débris* between the large masses of disintegrated gray substance of either side. The white matter retained more of its structural features than the gray; but nowhere could a normal fibre be seen. All showed a breaking up into larger or smaller droplets of myeline in some part of their course, or were the seat of varicosities of diverse sizes. The most striking histological feature of the cord at this level was the large extravasations of blood, which took up almost the entire gray matter and were numerous in the white. The vessels of the cord, especially those of the anterior horns, were enormously distended with blood. Considerable numbers of small spheroidal cells were present about the blood-vessels, both in the gray and white, but were not as numerous as might have been expected considering the lapse of time between the injury and death, and the consequent opportunity for inflammatory reaction. The central canal of the cord could not be detected. In its situation was a mass of blood and *débris*. Near the median line, far back in one posterior column, was a hook-shaped structure, consisting of cylindrical epithelium, derived undoubtedly from the central canal. This was the only representative of the lining of the canal that could be detected. The nerve-roots had suffered from the crush, some bundles of fibres being partially disintegrated. It cannot be said whether the anterior or posterior roots had suffered most. Numerous small extravasations of blood were present in the nerve-roots and in the pia. In the lowest part of the eighth cervical segment the topographical arrangement of the gray and white substance is much more nearly normal. The posterior horns are, however, too near together anteriorly, and too widely separated posteriorly, and no trace of the epithelial lining of the central canal can be made out. A few ganglion-cells present a normal appearance; most of them are atrophic. The majority of the fibres in the white substance are destroyed. In the anterior columns there are a few seemingly normal fibres. Hæmorrhages are numerous in the gray and white substance, but much less so than in the upper part of the segment. There is very little evidence of any inflammatory process, and collections of leucocytes are neither dense nor numerous. The majority of the nerve-roots appear normal, but one of the roots external to the cord presents the curious appearance of having preserved many normal fibres in its peripheral portion, while its central fibres have been disintegrated. In the seventh cervical segment the changes were of the same character as in the eighth, except that they were less in degree. In the sixth segment the evidences of mechanical disintegration are slight, but both the gray and white substances show considerable evidence of an inflammatory process. Considerable numbers of leucocytes are collected in the anterior horns, and in the connective-tissue septa of the white matter. The ganglion-cells are shrunken and without processes. There are hæmorrhages both in the gray and white substance, but they are small and few compared with those further down. At this level the posterior horns are still much displaced and injured, and the central canal does not exist. The topography of the cord gradually regains its normal features as we ascend to the fifth and fourth segments, but does not become perfect until we reach the third segment, in the lowest part of which the majority of fibres and ganglion-cells appear normal. Even at this level there are some accumulations of leucocytes about the vessels, and some small extravasations both in the gray and white substance, and the central canal is filled, though not distended, with blood. Above this level the cord soon becomes normal.

CASE II.—This case has already been elsewhere[1] recorded, but without the results of a microscopical examination. It may be very briefly summarized as follows: A man, aged 57, fell head first, a distance of twelve feet. On being brought to the hospital, he was found to be paraplegic below the arms, and to have paralysis of the intrinsic muscles of the hands. There were anæsthesia and analgesia below a V-shaped line across the upper part of the chest, and, roughly speaking, in the inner half of the arms, forearms and hands. Respiration was diaphragmatic. The knee-jerk, cremasteric, abdominal and plantar reflexes were abolished, and urine and fæces were



≡ Area of Anæsthesia and Analgesia.
○○○○ Area of Hyperalgesia.

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[1] Journal of Nervous and Mental Disease, January, 1890.

passed involuntarily. The paralysis spread successively to the flexors and extensors of the wrists and the triceps, and there was some loss of power in the biceps, supinators, pronators and pectoralis major muscles. The sensory loss remained stationary. The patient died after forty hours, and there was found to be a dislocation forward of the sixth cervical vertebra upon the seventh. The seventh and eighth cervical segments of the cord were much softened, and the left anterior root of the eighth nerve was torn across. The microscopical examination of the eighth, seventh and sixth segments of the cord makes it evident that the damage to the cord in this case was far less severe than in Case I., though the changes are essentially the same in character.

In the lowest part of the eighth segment there is only slight deviation from the normal outlines of the gray and white substance, but the majority of the ganglion-cells are atrophied and without processes. Many nerve-fibres, especially those of the posterior columns, are disintegrated, and the central canal, with its epithelial lining, cannot be recognized. There are numerous hæmorrhages in the gray substance, but the area occupied by extravasations is much smaller than in the cord from Case I. The hæmorrhages in the white substance are also relatively slight in this cord. Many of the fibres in the white substance have a morphologically normal appearance, but their axis-cylinders stain very imperfectly with carmine. In the upper part of the eighth segment the changes are more marked than in any portion of the cord. The gray substance is much broken up, the central canal is destroyed, hæmorrhages are extensive and numerous, and there are small collections of leucocytes about the vessels, especially those of the gray substance. Above this level there is a gradual return to normal structure. In the sixth segment there are many normal ganglion-cells, and the majority of the fibres of the white substance are unchanged. The central canal is greatly distended with blood.

The following case of crush of the upper cervical cord is presented with the permission of Dr. McCosh, who operated upon it. From a neurological point of view the history is somewhat imperfect, but it illustrates several important points.

CASE III.—Andrew Gunn, age 69 years; widower; plasterer. On July 12, 1890, while at work on an unfinished building, he fell three stories, striking his head and left side against a board flooring. Patient was found in shock, and taken to hospital. On admission, patient's condition was as follows: Temperature 97¾°. Pulse rapid and feeble. Respiration diaphragmatic and labored, the extraordinary muscles acting. Examination gives crepitus in the fourth cervical vertebra, over which region there is great tenderness. No information could be obtained regarding the existence of paralysis until several hours after admission, when the patient began to recover consciousness, and it was ascertained that all four extremities were completely paralyzed. Sensation was abolished below a line crossing the chest at the level of the third rib. No accurate observations were made of the area of lost sensibility or of the exact distribution of the motor loss, but the arms appear to have been completely paralyzed. The cremasteric, plantar, abdominal and patellar reflexes are quite abolished. There is incontinence of urine and fæces. An operation was thought to be indicated, and revealed a fracture of the lamina of the third cervical vertebra on the right side. The cord is said to have been badly crushed at this level, and the dura torn. The operation took place at 1.45 P.M., and death occurred at 7.30 P.M.

CASE IV.—Mary Jacobus, aged 35, fell from the fifth story of a tenement-house, through the dumb-waiter shaft, on Sept. 29, 1889. Examination shows transverse fracture of spine of scapula. Distinct retro-pharyngeal projection, causing some dyspnœa. Persistent pain over second, third, and fourth cervical vertebræ, increased by manipulation. Complete loss of power below neck. Anæsthesia below a transverse line three inches beneath clavicle. Total abolition of reflexes, both superficial and deep. Mental state clear. Retention of urine; incontinence of fæces. Temperature on admission 99°, subsequently varied between 101° and 105°. Respiration diaphragmatic.

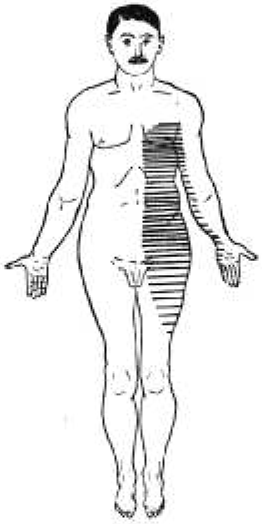
*Sept. 30th.*—Patient is unable to keep anything on her stomach; persistent vomiting. Marked distention of whole abdomen. The vomited matter is greenish, and much resembles the vomitus of peritonitis.

*Oct. 1st.*—There was a diaphragmatic spasm lasting several minutes, during which the patient became cyanotic. The motor and sensory paralysis continue stationary. A thorough examination made under ether reveals distinct crepitus over fifth and sixth vertebræ.

Operation decided upon. Operation at 3.15 by Dr. Briddon. Incision in median line from a point one inch below occipital protuberance to vertebra prominens. After cutting through the post-vertebral muscles, the right lamina of the fifth cervical was found freely movable, and was removed, together with the spinous process of the same vertebra. The left lamina of the sixth vertebra and the corresponding spinous process being depressed, both were elevated and removed. The connective tissue lying upon the dura mater was gently lifted, and a small longitudinal rent could be seen in the dura mater, through which the substance of the cord protruded. The cord at this level was contused and softened, and there was a free escape of cerebro-spinal fluid. The wound was dressed, and two superficial sutures were put in place to approximate the margins of the wound and hold the gauze in place. An aseptic compress was put over this and a plaster-of-Paris jacket bound over the whole. The jacket extended from the parietal eminences to the lumbar region, and was made to conform to the shape of the body. The patient was put to bed and stimulated; but she became delirious, and died at 9.45 P.M. Temperature immediately after death, 108°.

*Autopsy.*—Considerable congestion of peritonæum. Intestine tympanitic. Ecchymotic spots one inch in diameter in peritonæum of ascending colon. Body of sixth cervical vertebra is slipped a little forward on the fifth. The cartilages on both sides of it are crushed, and there is a sagittal fracture through the body. The cord is crushed, softened, and a little hæmorrhagic at this point. The fifth and sixth spines and parts of the laminæ were removed.

CASE V.—J. S., aged 67, fireman, has always been in good health. On the morning of Feb. 9, 1891, he fell from the head of a stairway a distance of twenty-two steps, and when picked up soon after was in a dazed condition. He had been drinking the night before. Soon after he fell the patient was admitted to the hospital. On admission the patient's temperature was 99°; pulse 80, full, regular; respiration slow and regular. He suffered at this time from acute pain in the region of the sixth lower cervical spines. No fracture or displacement of the vertebræ could be made out. Over the fifth, sixth and seventh vertebræ there is great tenderness on pressure. An examination, made during the afternoon of the 9th, shows that the flexors and extensors of the wrist are all but paralyzed on the right side, and that the flexors and extensors of the wrist of the left side are much weakened. The intrinsic muscles of the right hand are quite powerless; those of the left hand are only partially paralyzed. On the trunk, extending as high as the nipple on the left side, is a large area of altered sensibility, in which there is slight tactile anæsthesia and entire loss of sensibility to pain and temperature. These changes in sensibility exist both in front and behind, and extend to within one inch of the median line. As one passes over the trunk toward the lower extremity, the analgesia grows gradually less marked, and fades out into normal sensibility on the thigh in front and upon the buttock behind. The condition of sensibility is represented in the accompanying diagram. It will be noted that the diminished sensibility



extends along the inner border of the arm upon the hand. Nowhere upon the arm was sensibility entirely absent, and the anæsthesia (to touch, pain, temperature) fades into the normal before the median line of the arm is reached. The lower extremities were repeatedly examined with reference to their power, and at no time could any loss be made out. The reflexes were all normal, except the knee-jerk of the left side, which was increased. There was also ankle-clonus on the left side. On the morning after admission there was marked tympanites and the abdomen was tender to pressure. The distention increased very much during this and the following day, and the patient vomited dark greenish matter repeatedly. The temperature remained normal. In the course of three or four days the distention subsided.

The anæsthesia remained as described for about three days. Then it grew gradually less pronounced, until, at the end of ten days, sensibility was normal on both sides of the body. The anæsthesia disappeared first from the arm. There was an improvement in power, but the recovery was partial only. Seventeen days after the accident the intrinsic muscles of the left hand had recovered power and the extensors were much improved. On the right side there was slight improvement in the flexors and extensors of the wrist, but scarcely any in the intrinsic muscles of the hand. The forearms were not measured, but it is believed that at the end of seventeen days there was more atrophy than could be accounted for by simple disease. The muscles of the forearm, especially the flexors, became very flabby. The atrophy of the interossei was very distinct. Within the first week an electrical examination was made. No changes could be made out in the muscles until ten days had passed, when there was slight diminution to faradism in the flexors and extensors of the forearms. Subsequently there was very little change in this respect, the diminution in faradic irritability remaining slight. The first examination with galvanism was made on the sixteenth day, and showed simple diminution of irritability in the muscles that were the seat of diminished faradic reaction. Nothing positive can be said concerning the electrical state of the interossei. Two days after the injury, ankle-clonus could not be obtained on the left side, and on the fourth day the knee-jerk seemed no longer increased. The patient was heard from six weeks after this was written and is said to have received full power in the arms.

*Observations.*—The case just recited differs in kind from the first four cases whose histories have been read, in that it is an instance of relatively slight damage to the nervous structures of the cervical region. As regards the localization of the damage, certain things are very clear and others are equally obscure. The loss of power limited to the forearms and hands can be explained only on the supposition that there is a bilateral nerve-root lesion, involving, in all probability, the roots of the sixth, seventh and eighth nerves. The damage to these roots must be slight in degree, as there was no evidence of even commencing degenerative electrical changes in the muscles at the end of seventeen days. The peculiar distribution of anæsthesia upon the trunk and left arm cannot be satisfactorily explained. The limits of the lost sensibility upon the chest and arm of one side recall the area of loss in the first and second cases of this paper, and suggest that pressure upon the right half of the cord may be responsible for it. The objections to this view of the case are, first, that there is no loss of power in the leg of the side of this supposed cord-lesion, and, secondly, that there was for a time an increase in myotatic irritability on the side of the anæsthesia. The first four cases that have been recorded are all examples of severe crushing of the spinal cord, and have many clinical features in common. They differ somewhat in the localization of the cord lesion, to which their symptoms are due, but may nevertheless be regarded as constituting a distinct clinical group—complete cervical paraplegia from sudden total transverse destruction of the cord at a certain level. Many of the more striking symptoms of these cases are already sufficiently well known, and these will not be discussed. But there are other features of this class of cases that are less well known and understood, and it is especially to these that attention is invited. Among the most interesting features of these particular cases are their bearings upon the localization of the functions of the cervical cord. In Case I. there was an upward extension of the motor paralysis, from the interossei and flexors of the fingers to the extensors of the fingers and wrists, the pronators and supinators, the triceps, biceps and deltoids successively. In Case II. the order of advance was much the same, namely, intrinsic muscles of the hand, flexors of the wrist, extensors of the wrist, pronators, supinators and biceps. In both cases the anæsthesia occupied very nearly the same regions, namely, the body and legs below a V-shaped line across the upper part of the chest and the inner half of the arms, forearms and hands. While the motor symptoms progressed upward in both cases, the area of anæsthesia made no advance in the first case, and very little in the second. When the patients were first seen they presented essentially the same motor and sensory phenomena, namely, weakness of the hand-muscles and the distinctive anæsthesia above mentioned. The cord-lesion was essentially the same in the two cases—that is, complete crushing of the eighth segment and partial softening of the seventh and sixth segments. We are justified in attributing the peculiar distribution of anæsthesia to the crush of the eighth segment. In both cases the bone-lesion was a fracture-dislocation of the sixth upon the seventh vertebra. In three of the four cases there was pressure of the displaced or fractured vertebræ upon the cord at the time of operation or autopsy; in one case (Case II.) there was very slight displacement only, at the time of the autopsy, a degree of displacement quite inadequate to account for the damage sustained by the cord. We are quite justified in believing that in some cases of crush there is merely a momentary displacement of the bones, followed by a springing back into place or nearly into place. Of course, in these cases the original displacement is readily reproduced at the autopsy or even during life.

In all total transverse lesions of the cord, and especially in those of the cervical and lumbar enlargements, certain symptoms are referable to the damage of the cord as a central organ, as opposed to those symptoms which depend on the obliteration of the functions of the cord as a conductor of impulses. These symptoms in the cervical region include loss of power and cutaneous sensibility, muscular atrophy and degenerative electrical reactions. It is more than probable that in Cases I. and II. the loss of power in the hands and the distinctive distribution of the lost sensibility in the arms are referable to the blotting out of the cord's central functions in the eighth cervical segment. That this is true of the loss of power in the hands is made certain in the case of our first patient by the fact that changes in the electrical reactions of the hand-muscles were detected shortly before death. A slight degree of atrophy also was apparent in the hand-muscles (interossei especially), and both the electrical reactions and the atrophy are surely dependent on damage to the lower segment of the motor path—damage located in this instance in the eighth cervical segment. But the electrical changes just spoken of are of considerable interest in another connection. The patient in whom they were observed lived a little less than seven days from the time of the accident. An electrical examination was made on the fifth day as a matter of routine, and with no expectation of finding any alteration in the irritability of the paralyzed muscles. There was found not only greatly diminished faradic irritability in the hand-muscles, but entire loss of galvanic contractility, with the exception of one interosseus in which the irritability was diminished and associated with equality in the polar reactions. On the sixth day there was entire loss of faradic and galvanic irritability in the hand-muscles and distinct change in the forearm muscles. The nerve-reactions were not taken, and the currents used were unfortunately not measured, but both the galvanic and faradic were extremely painful and produced violent contractions in normal muscles. The observations were several times repeated each day, in order to exclude all sources of error. We are justified in attributing these alterations in muscular irritability to commencing degenerative changes in the nerves in question—changes of an intense and rapidly progressive character. It is interesting to note the entire loss of galvanic irritability in association for a short period with slight preservation of faradic contractility. It is also of interest to observe that no reversal of the polar formula was detected, although in some muscles there was a polar equality and distinct sluggishness of contraction.

The development of electrical changes in this case was considerably more rapid than any that have come to my notice. It seems not improbable that there may be some relation between the rapidity of the degenerative changes, which we may safely assume to have existed, and the severity of the lesion, than which there can be no greater.

In two of the cases above reported (IV. and V.) there were abdominal symptoms worthy of note. In each case, on the day after admission, the abdomen became tympanitic, exceedingly tender to pressure, and repeated vomiting occurred, the vomitus having at one time a dark greenish color. The abdominal distention became very great, but began to subside, together with the pain and tenderness and vomiting, in the course of a few days. What these symptoms—which for a time suggested a developing peritonitis—are due to, we cannot venture to suggest. In Case IV. the autopsy revealed the presence of considerable congestion of the peritoneum, marked tympanites, and numerous ecchymotic spots in the peritoneum of the ascending colon. Symptoms similar to these have been observed by me in two cases of injury to other regions of the cord. In one case there was crush of the cord in the lower dorsal region; in the other, the symptoms followed directly after an operation in the lumbar region, done many months after a hæmorrhage into the cauda equina. It is believed that other observers have not noted these symptoms.

The temperature in cases of injury of the cervical cord varies much, according to the severity of the damage. In the cases of slight injury, like Case V., the temperature may be either normal throughout, or may be slightly elevated at first. In the severe cases of crush of the cord, the temperature varies within wide limits, and apparently without much reference to the exact level of the injury. In each of the four cases of severe crush of which the histories have been recited there was a sharp rise in temperature during the first twenty-four hours. The temperature rose in one case to 105.2°, in another to 104°, within a few hours of the injury. In the cases where the patients survived the accident several days, the temperature fell from the maximum of the first twenty-four hours, in one case to normal, in another nearly to normal, to rise once more just before death, when the highest temperature was attained.

Similar temperatures have been observed in cases of injury, or operation, in the lower dorsal and lumbar regions.

It seems probable that the considerable elevation of temperature observed in these and other cases of the same character (Thorburn's cases) is due in some way—at present obscure—to the destruction of nervous tissues, since the rise is absent or slight in cases of slight injury, no matter what may be the degree of shock or bone injury.

The last feature of these cases to which it is desired to call attention is the state of the reflexes, especially the state of the knee-jerks. The superficial reflexes, cremasteric, plantar and abdominal, are commonly lost from the beginning in cases of complete crush of the cord, but a day or two sometimes passes before they are all lost. If the patient survives for several days or a week, some or all of the superficial reflexes return, as in Case I., where the cremasterics returned on the fifth day. The abolition of these reflexes is not invariable in severe crush lesions. In slight lesions of the cord there is ordinarily no change in them.

Far more important than the condition of the superficial reflexes is that of the knee-jerks. In all four of the cases of complete crush of the cervical cord, the knee-jerks were abolished, from a very early period after the accidents which caused these crushes, respectively. In all four cases the motor and sensory paralyses below the level of the lesion were complete; in all, the termination was fatal. Two of the cases, it is true, were operated upon; but it is absolutely certain, from the nature of the damage found, that these cases would have terminated fatally if left to themselves.[2] Thorburn has reported a large number of cases of damage to the cervical cord, some severe, some slight. In ten of these cases the knee-jerks were lost immediately after the injury was received. In all ten, the motor and sensory paralyses below the level of the lesion were complete; in all, the termination was death. On the other hand, in a large number of cases in which the motor and sensory paralyses below the level of the injury were partial in degree or distribution, and in some of which there was recovery, there are none in which the knee-jerks were lost early. We may safely conclude, therefore, that *when bilateral loss of knee-jerks follows immediately upon a sudden lesion of the cervical cord, we have to deal with a case of extensive or total transverse damage to the cord,* and that the patient *will die from it.* It is not urged, of course, that this diagnostic and prognostic sign be used to the exclusion of other associated conditions. Immediate complete loss of motion and sensation below the level of the lesion are equally important indications of the severity of the lesions, and should be taken in conjunction with the state of the knee-jerks when this is practicable. In cases where the patient is unconscious, we must rely on the state of the knee-jerks alone, at least for a time. It must not be thought that loss of knee-jerk is confined to lesions of the cervical cord; it occurs in severe crushes of the dorsal cord as well.

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[2] A Contribution to the Surgery of the Spinal Cord, 1890.

These considerations lead to one important practical conclusion, namely, that cases of crush to the cord presenting complete or considerable paralysis of motion and sensation below the lesion, and associated with loss of knee-jerks, should not be operated upon. It would not be necessary to state so self-evident a fact but for the reason that surgeons sometimes operate on these cases "to relieve pressure," and in the hope of some day finding a case which will recover. At the present day we are able to distinguish between nerve-root lesions and crush lesions of the cord, and should limit operative procedure to the former set of cases. This does not apply to cause of simple pressure on the cord, or to partial crush with fracture or dislocation.

A phenomenon so remarkable as loss of knee-jerk from a distant cord-lesion is of no little theoretical interest. How can the abolition in these cases of a reflex dependent on the lumbar cord be explained? We cannot hope to give a fully satisfactory explanation, but we may perhaps clear up the ground to such an explanation.

Thorburn[3] attributes the loss of knee-jerk to the depressing action of shock upon the reflex centres, and likens it to a case of temporary retention of urine, after an operation for genu valgum. This seems a very unsatisfactory explanation. Granting that shock is capable of abolishing reflex action, we must hold that it only rarely acts in this way. Most of the cases of severe concussion of the entire cerebro-spinal nervous system show no abolition of the knee-jerks; in the cases that do show it, the loss is evanescent. Again, many cases of slight injury of the cord are associated with the severest shock, and in none of these cases, so far as we know, has the knee-jerk been abolished. The duration of the effects of shock must be admitted to be short. What do we find to be the facts in our cases of severe crush? Does the knee-jerk return, and if so, when? In one of our cases the injury was survived seven days, but there was no return of knee-jerk, and there were no other evidences of shock. Thorburn states that in one case the knee-jerk returned in five weeks; but a careful reading of this case makes it questionable whether the knee-jerk was ever really lost. But, admitting that the knee-jerk may return after several weeks, we must still insist that the date of return is much too late to make shock responsible for the original loss.

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[3] Injuries to the Cervical Region of the Spinal Cord. Brain, 1887.

Bastian,[4] who also has called attention to the loss of knee-jerk which follows total transverse lesions of the cord, believes that loss to depend on a shutting off, by the lesion, of reflex-exciting influences passing downward from the cerebellum through the gray substance of the cord. In support of this view, a case of Thorburn's is quoted in which the knee-jerks were lost and autopsy showed a lesion (hemotomyelia) chiefly of the gray substance. The idea is that in this case the reflex-exciting cerebellar influence was cut off by the lesion of the gray substance, while the supposed reflex-depressing influence continued to pass from the cerebrum by the white substance. This ingenious idea will not stand when we look carefully into the character of the case which is supposed to support it. Speaking of the findings in this case, Mr. Thorburn quotes the following: "This hæmorrhage (constituting the hematomyelia), which measured in its vertical extent from one and one-half to two inches, 'was in the greater part of its extent situated centrally, occupying the whole of the central gray matter, and extending but little into the white substance, which in its neighborhood was merely softened and of faintly yellow tinge.' The white substance was 'merely softened,' but we consider this an excellent reason for excluding the case entirely, as an evidence of the correctness of the above-mentioned hypothesis."

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[4] British Med. Jour., March, 1890.

What can we substitute for these explanations? Briefly this: Whatever hypothesis we employ to explain the loss of knee-jerks, we must lean upon the idea of inhibitory influences from distant parts of the central nervous system, since the lesion in these cases is certainly nowhere in the reflex arc. We cannot throw any light on the nature of this inhibition, but we may indicate its source and character. Its source is the upper end of the undamaged lower portion of the cord.[*] In character it is irritative; that is, the result of active, irritating, pathological processes in the nervous structures. The evidence that the source of inhibition is in the upper end of the undamaged cord is that in some of our cases the parts above this were, for a short distance, completely disintegrated, no normal fibres remaining. The influence cannot, therefore, come from above the lesion. Immediately above and below this lesion the disintegration is less complete, and there are inflammatory changes—inflammatory changes which, though less than might have been expected, are nevertheless sufficient to produce over-action in adjacent healthy tissues. On *a priori* grounds, therefore, we might expect to find a considerable degree of over-action from irritation dependent on the parts above and below the crush. But we have additional evidence of the best kind that this is in reality so. From the upper end of the uninjured cord we can, of course, expect no definite symptomatic evidence, as we have complete paralysis of motion and sensation below the lesion. From the lower end of the upper uninjured region of the cord we get the evidence to make our case complete.

There were certainly in two of our cases, and probably in all, symptoms of an intensely irritative kind, referable to the lowest part of the upper end of the cord, namely, great pain in the arms just outside (above) the border of anæsthesia in one case, and intense girdle pain about the neck in another. Powerful contraction of the biceps also was present in one case, and this did not disappear until the paralysis ascended, from ascending inflammation of the cord. Have we not, therefore, excellent reason to assume that a similar irritative influence is exerted from just below the lesion on parts below? When we say the loss of knee-jerks is due to irritative inhibition, we are using a legitimate expression, if we use it in the sense here given, and one which indicates probably truthfully, though vaguely, the character of the influence that gives rise to this most interesting phenomenon of loss of knee-jerks.

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## ATROPIN AND DUBOISIN AS SEDATIVES AND HYPNOTICS.

Dr. N. Ostermayer, of Budapest (Allg. Ztschr. f. Psychiatr., xlvii., 3 and 4), comes to the conclusion that atropin, used subcutaneously, in doses of 0.001 to 0.002 grammes, in the insane, exercises a sedative and indirectly hypnotic action, lacking, however, the certainty and strength of the action of hyoscin. According to him, it does not cause collapse. The patients accustom themselves to the remedy. A disagreeable side action was observed in only one case, where diarrhœa and vomiting followed its administration. Atropin often acted where morphine and hyoscin failed. The sulphate of duboisin acted, injected subcutaneously, in doses of 2 to 3 mg., as a prompt and intense sedative. In insomnia, without motor restlessness, 1 to 1.5 mg. also caused sleep. No disagreeable symptoms were noticed during its use. Its action diminishes after some time. The writer doubts the assumption of Ladenburg that duboisin is identical with hyoscyamine. He recommends duboisin instead of hyoscin, especially in cases where diseases of the heart and blood-vessels render the administration of hyoscin dangerous; also its cheapness recommends its use. P. and P.